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March 30, 2020

Brett Reynolds, Esquire
and Associates PC
1250 Northeast Loop 410
Suite 310
San Antonio, TX 78209

RE: Morgan Harris-Workman
DOB: 12/19/1997

Dear Mr. Reynolds:

At your direction I have evaluated Morgan Harris-Workman with regards to possible neurologic injuries which may have been sustained by her on November 5, 2017 when she was one among many wounded in a mass shooting at her church. In pursuit of that I have reviewed the following records: Wilson County EMS; hospital ED records from Connally Memorial Medical Center and University Hospital; office records and reports from S. Gazda, M.D., S. Shriner, M.D. and R. Nieman, M.D.; and expert report from C.Ticknor, M.D.

I examined Ms. Harris-Workman on March 11, 2020. She explained that when the gunfire erupted, she was seated at the audio control panel, separated from the rest of the congregation by a low partition through which she was told the projectile(s) probably passed before striking her in multiple places. She said that she was instantly aware of being struck in her left leg but not immediately aware of other injuries. She said that she remained conscious and, though aware of bleeding, was able to ambulate, probably impaired only by pain in her left leg. That she was able to assist others more severely injured than she and that she didn't fall while doing so reinforces her report that she sustained no unconsciousness and had initial gross impairment of ambulation. Her own initial care at

Connolly Memorial Hospital involved cleansing of her multiple wounds and initial imaging before she was then referred from the local emergency room at to a tertiary care center for more definitive assessment and further imaging. She said that she understood that it was ultimately determined that she had sustained multiple shrapnel injuries to the left thigh and calf of which one fragment was proximate to the left knee joint and that there were also fragments of shrapnel lodged in her right shoulder and the right side of her face. The final determination was against debridement or attempt at direct shrapnel removal owing to the multiplicity of small fragments being widespread in the surrounding tissues.

Ms. Harris-Workman said that in the weeks immediately following her injuries she had diffuse swelling of the distal left thigh and the entire left lower leg and she opined that any difficulty she experienced walking at that point was explained entirely by pain, accentuated on weightbearing and confined to the left lower extremity. Over a stretch of about four weeks she said that the pain and swelling subsided and that she became progressively more ambulatory, initially on crutches, and finally, during a subsequent additional stretch also of about four weeks, with a steadily declining and ultimately ending dependence on the use of a cane. She said that she was not aware of either motor or sensory disturbance in either lower extremity during approximately the initial eight or ten weeks following the gunshot injury.

In retrospect, she said that her first awareness of gait disturbance unrelated to pain was about at the time of her wedding, roughly ten weeks after the initial injury, but, at that time she attributed what seemed to be a problem involving both feet to residual swelling though the swelling had been present in the left leg only and had not seemed to involve her feet. Over the next approximate six to eight weeks she experienced an increasing gait difficulty because of bilateral foot dragging and that steady worsening problem with walking became determinative of her seeking medical attention. She described herself particularly vulnerable to tripping because of foot drop which seemed to involve the right foot even more than the left. She began to reuse the cane which had been abandoned several months before. At some point, she also became aware of decreased sensation in both feet, right foot more than left. While she can approximate the onset of motor deficit from roughly the time of wedding, she is less clear about the onset of the deficit of sensation except to say that her general sense is that motor deficit preceded her experience with diminished sensation to touch and diminished awareness of cold or heat of the bathroom or bathroom shower floor, right foot more than left and on the lateral aspect and sole of either foot more than the medial and upper surfaces. She did not experience similar motor or sensory symptoms in her hands.

Ms. Harris-Workman estimated that the course of motor deficit thereafter worsened more slowly over a subsequent period of approximately six months, finally plateauing at about 8 or 10 months after the onset of the gait disturbance or approximately one year to 15 months after her initial gunshot injury. Whether and to what degree attributable to chelation, she stated that her motor and sensory deficits have both improved over the most

recent year, doing so in the reverse sequence of that experienced in the onset or, in other words, with sensory deficit improvement occurring before improvement in motor impairment, and improving to a degree inverse to the relative magnitude of maximum impairment or, in other words, with sensory improvement being greater than motor improvement. Fairly early in the sequence of neurologic evaluation and treatment, which began some weeks after the onset of the gait disturbance, she was fitted for MAFFOS that she wore for about six months but were later discontinued in favor of a device to facilitate foot dorsiflexion. At present she describes herself as partly ambulatory without braces although limited in ability by time-and-effort-dependent fatigue. She that she operates the brake and gas petal of her car by using her "whole foot," as she describes it, apparently to bypass her weakness with forefoot flexion, instead of the usual maneuver. In a subjective sense she still experiences weakness at her right foot/ankle more than her left and, although she is constantly aware of her persisting motor deficits, she is not so continuously aware of sensory deficits which, by contrast with the motor deficits, she said seem to wax and wane although generally to effect the right foot more than the left and the lateral regions of either foot more than the medial and to have associated variable amounts dysesthetic sensation when touched. She also described ongoing intermittent discoloration of both distal lower extremities still evident, provoked especially by cool ambient temperature or change in temperature.

Ms. Harris-Workman said that after the injury she was not initially aware of cognitive symptoms but has become more aware of such over roughly the past 12-18 months. In this regard she does not offer a clear history of evolving deficits so much as one of increasing awareness that she now struggles with multiple tasks which imply she is now experiencing trouble with cognitive function. Most obvious is that she acknowledged that she has become "forgetful." By that expression she refers to a broad array of struggle with tasks for which memory may be implied as a part but not all of the cognitive task requirements. For example, she spoke of difficulty retrieving phrases, expressions or specific words. She said that she has been troubled by remembering directions, appointments and specific details of directions and appointments but admitted that the problem has been further compounded by her inability to find a successful system to compensate or to prevent missing the details. At one point she commented, "If I don't get something in there (gesturing to her Smart phone) then I can completely forget…" This group of problems that she calls "forgetfulness' is now leading to increased frustration at work. She offered a specific example of a now frustrating work task which previously had not been an area of struggle. She now finds herself making mistakes and needing correct, revise or re-work.

Ms. Harris-Workman acknowledged growing up with less interest in reading than her siblings, described by her as "bookworms." That said, however, she never, until the recent 12 or 18 months, experienced an actual sense of struggle with reading. Now she finds herself needing to read some types of material, work material especially, 6 or 7 times and, even then, she said that she is no longer confident in understanding or remembering what she just read. In a social setting she said that she has now lost her ability to "multi talk," by which expression she means her ability to be involved simultaneously in multiple

conversations as would be required to jump back and forth from one conversation to another in a large family gathering. Whether it reflects greater difficulty with organization or memory or some combination thereof she is unsure, but the functional consequence of her difficulties in the last 12 to 18 months is that she now has problems managing her bills. When asked about specifically about her attention to driving, a particular concern in face of her commuting time of approximately two hours daily, she acknowledged that she has had experiences in recent months in which she has felt less confident about whether she has maintained awareness of the immediate traffic situation and/or her immediate location in her travel sequence. These experiences notwithstanding, however, she has had neither traffic accidents nor documented traffic violations.

She explained that she grew up in a rural area near the San Antonio, Texas, living there for her first 8 years before being briefly in Montgomery, Alabama and then returning to Texas at age 12 years. She said that she was homeschooled throughout and that she never took any specific standardized testing as none was required at that time. She said that she never had subjective sense of difficulty with a particular academic subject and she recalled no remarks from her mother that might hint at such. She said that the IT skills employed in her current job have been self-taught

Her prior medical history is benign. She has no history of known lead or other toxic exposure, whether in household, through travel or because prior work or hobby interests. She has no known history of prior focal or specific neurologic deficits. When asked about her understanding of the conversations in her chart regarding Chiari malformation, she reaffirms the implication of that disorder having been asymptomatic thus far. She has no experience with any type of prior neurologic symptomatology involving unconsciousness, altered memory or concentration or any difficulty with gait, balance, strength or sensation. She did, in early adolescent years, experience pain or stiffness on neck rotation. That was evaluated at that time with no cause identified and the problems resolved spontaneously.

She reported that her father is approximately 57 years of age and her mother, approximately 52 and said that she is the youngest of four children. She is not aware of any specific neurologic condition other than a paternal grandmother with some form of dementia, possibly Alzheimer's. The only family medical predilection which she is aware is that of hypertension (her father and paternal grandfather) and atherosclerotic heart disease (her paternal grandfather).

On examination she was easily engaged and used fully intelligible speech. There was no obvious evidence of expressive or receptive language deficits. While she conveyed a clear sense of intent to perform well, she quickly became frustration when she appeared to perceive herself doing poorly. For example, when given written computations she made lots of stops and starts and verbalized comments of frustration and self-deprecation. In the end her written computations were poorly done. Writing in response to a standard dictated sentence, she made one omission–spelling error and two full word omissions. She identified coins by denomination and value but, despite using an organized approach, she

ultimately was not accurate in summing the total. Asked to read a standard high school level paragraph, she did so with minor hesitancy and one substitution error (neither visually nor phonetically similar to the printed word) and, although she did retrieve concrete information from the passage, she had considerable difficulty extracting any inference. She did poorly with informal computations utilizing sequences of days in the week or months in the year and, likewise, did poorly with serial subtractions of the same number. There also she interjected comments implying frustration ("I don't know where I was…") and appeared to lose her place as well as to make careless mistakes. She frequently seemed aware of making mistakes and tried to regroup but was unsuccessful.

She displayed an overall healthy and nondysmorphic appearance but showed obvious mild distal muscle wasting in both lower extremities. She had fully intact visual fields and full, conjugate extraocular movements with brisk OKN responses, normal pupillary responses to both modalities and fully intact cranial nerve functions. Funduscopic examinations were not performed. Tests of cerebellar function are well executed by the upper extremities with no display of adventitial movement or dysmetria. Repeated toe tapping was slow and awkward. In routine walking she displayed a mild-to-moderate steppage gait, had a limited ability to walk on her forefeet but was completely unable to either walk on her heels or to execute tandem gait. Her standing balance was secure only on both feet held somewhat apart. Sensory examination showed mild" glove and stocking" diminution to perception of both pinprick and cold in all extremities, subjectively more pronounced in the right foot, especially lateral aspect. In both feet sensory disturbance was more marked on the lateral and plantar aspect. Appreciation of joint sensibility was clearly intact in both upper extremities and the left lower and probably intact in the right lower (two errors in eight trials but no errors in exam of the other extremities). There were no observed muscle fasciculations. Motor strength was good throughout with the exception of mild (4/5) distal weakness in the lower extremities, particularly involving extensors of the great toe and distal foot more than inverters of the feet and flexors of the ankle (minimal involvement) with mild but distinct accentuation on the right side compared to the left. There was no palpable nerve enlargement. Reflexes were intact in the upper extremities but only trace present at the brachioradialis. Right knee jerk required reinforcement and both Achilles reflexes were absent. Abdominal reflexes were present but weak.

In sum, her examination findings are notable for mild general cognitive impairment spanning multiple cognitive domains and accentuated by tasks requiring attention to detail and for clinical findings of a distal neuropathy, primarily involving the lower extremities, with motor deficits significantly greater than sensory. She also appeared to have a vaguely defined diminution of sensation on the left trunk.

The above referenced medical records parallel Ms. Harris-Workman's report, and indicate acute findings of multiple acute shrapnel wounds, documented both by both examination and by radiograph, involving especially the distal left thigh and proximal left lower leg but with additional scattered shrapnel scattered in soft tissue surrounding the right shoulder area and in the facial area of her right cheek. Neurology records further reinforce a

subsequently evolving subacute progressive deficit, clinically and electrographically consistent with a peripheral neuropathy involving motor much more than sensory function, beginning some months after her initial gunshot injury and progressing over at least 9, possibly 12 months thereafter with later partial resolution to approximately the present point. On comparison of the findings today with those of the available medical record, the neuropathy has been stable for approximately the last 9 to 12 months

The medical records of this case document a thorough exploration of the differential causes of the neuropathy and an appropriate conclusion that it is a toxic neuropathy attributable to lead poisoning from retained bullet shrapnel. The records further show that she was placed on a diet with appropriate restrictions and supplements, especially calcium, and that she underwent chelation which corresponds roughly with the time of stabilization and subsequent modest motor improvement. All these points are roughly parallel to the historical narrative, above, given by Ms. Harris-Workman at the time of this evaluation. Of note is that the available laboratory studies in this file do not document any of the additional but common nonneurologic abnormalities sometimes associated with lead poisoning although those are usually associated with more pronounced elevations of blood lead level than are recorded here.

These medical records are also significant for the report of a seemingly incidental finding of Chiari 1 malformation in which there is combined hydrocephalus and a cervical spinal cord syrinx. According to the records this process has been asymptomatic to date, a point reinforced by both her history and by the exam findings today, although the above noted vaguely outlined truncal hypesthesia to pinprick may possibly indicated that it is now not without demonstrable abnormal finding. At this writing, I have not personally reviewed the neuroimaging which lead to the Chiari 1 malformation diagnosis as there is a practical problem created by the current pandemic and the considerable impact on obtaining such material. However, reserving the right to readdress that at a later point, I accept the Chiari type 1 malformation diagnosis under advisement but consider that matter largely if not entirely unrelated to issues of litigation in this case. However, I am unable to completely dismiss from analysis the Chiari type 1 malformation diagnosis because of the presence of cognitive impairment, discussed below and attributed to lead intoxication, as that diagnosis might conceivably surface in future decisions about management even if it is by all medical reasoning not directly connected to lead poisoning.

Absent from these medical records to date is any information about the cognitive abnormalities described historically by Ms. Harris-Workman and documented on my exam, above. The omission likely has a simple basis, explained first on one hand that Ms. Harris-Workman conveys a strong sense of "blaming herself" rather than seeking medical attention about her "forgetfulness," as she termed it. The affected individual first denying and then blaming himself or herself, especially for any cognitive struggle, is both a well-known and widely encountered barrier to care. To some extent, however, there are hints given by Ms. Harris-Workman that, in speaking of frustration, she might be relating to her cognitive struggles in her recent psychiatric evaluation. The second explanation for the

omission from the medical record of information about cognitive issues is the apparent focus by the various medical providers on the patient's "chief complaint," of difficulty waking. It is generally the standard of care in a medical evaluation to first address the problem for which the patient seeks care and that was clearly well done in this case. Nevertheless, for reasons set forth below, omission of exploring the possibility of cognitive impairment is unsatisfactory in the setting of possible lead toxicity.

Although historically man's use of lead is probably predates civilization itself, the appreciation of its toxicity dates mostly to the last two centuries and, despite much knowledge about some of the aspects of that toxicity, huge voids in understanding persist. There is no lead normally in the human body for, unlike other metals such as calcium, iron, zinc, magnesium or manganese, lead is not incorporated normally in any known metabolic process in humans. Although the presence of any lead is abnormal, plasma levels are the most available means of directly documenting prior exposure and facilitating some medical and environmental decision making even though the plasma lead levels, especially in the lower ranges, don't correlate quantitatively very well with the extent or degree of injury, especially to the central and peripheral nervous system, which are selectively organs most vulnerable to the toxic effects of lead.. Although lead is a known toxin to both central nervous system (especially in the young) and peripheral nervous system (especially in adults?) there is not general agreement on how that toxicity is brought to fruition as there is evidence for multiple mechanisms involved. While much public health and environmental medicine has focused, and still does, on sources for and mechanisms of gastrointestinal absorption (especially in children) or alveolar absorption (especially in adults), absorption from lead containing foreign bodies and bullets is also now unfortunately widely documented as well. All three means probably depend mostly on subsequent transport of lead, mostly bound to plasma proteins, to reach organs vulnerable to the toxicity, of which the central and peripheral nervous system seem uniquely so. The plasma flux is dynamic and time limited and the subsequent evolution of clinical manifestation requires time for evolution, especially the central and peripheral nervous system, but the effects of damage to the nervous system recede only partly as the plasma flux wanes as lead becomes somewhat sequestered. This lingering damage, an aftermath of prior exposure, also distinguishes central and peripheral nervous system lead toxicity from that in some other vulnerable organs such as bone marrow, for example. The present case illustrates all of these puzzling points. The latency after initial lead exposure, the subacute progression of the resulting neuropathy, a very limited response, if any, to chelation (which can only draw off the unbound plasma fraction), a modest recovery and a significant residual deficit all match the known picture of the neuropathy of lead poisoning. The highly toxic effects on the central nervous system, especially on the young, have become somewhat clear only in the modern era although are now cited by historians as possibly contributory to the decline of Roman Empire since lead was used to sweeten cheap wine, which, if correct, may illustrate widespread central nervous system toxicity in more mature individuals. That is relevant in this case for some have argued that a degree of central nervous system deficit (cognitive impairment) probably accompanies peripheral

nervous system involvement (neuropathy) in almost all cases and, therefore the possibility of coexisting central nervous system deficit should be explored and perhaps considered supportive evidence in cases where peripheral nervous system deficit (neuropathy) can be attributed to lead toxicity. In short, the medical record and history are clear as to lead poisoning being explanation for the abnormal central and peripheral nervous system findings detailed above.

However, the limitations to understanding the pathophysiology of lead toxicity create difficulty for specifying the long-term prognosis. In this case it is reasonable to assume that transport of lead by plasma, rather than direct tissue contact, would be the primary vehicle by which lead indirectly released from bone or directly released from retained shrapnel would pose future risk to distant organs, primarily the central and peripheral nervous system, given the likely low plasma levels involved and the heightened vulnerability of the nervous system to the toxic effects of lead. The potential for future flux of sequestered lead occurs is well known and is easily exemplified by the numerous reports of clinical lead poisoning, including neuropathy, after long periods of latency following lead exposure, including that arising from gunshot wounds. Despite limited understanding of all the likely factors which might lead to a future flux of lead, two special circumstances are especially germane to this case. The first concerns the mobilization of previously sequestered lead in association with pregnancy. The well documented mobilization of lead in late pregnancy and the early postpartum periods pose special threat because plasma lead is readily exchanged across the placenta and because there is known increased vulnerability to lead by the immature brains. Based on current knowledge, therefore, the retained lead in shrapnel and the lead sequestered in bone from prior plasma flux pose dire risk for fetal brain damage from lead and thus advice is given to Ms. Harris-Workman that avoid pregnancy. The second special circumstance posed in this case arises from the known proximity of several shrapnel fragments to the left knee joint and the known "mobility" of missile fragments which could migrate into the nearby knee joint capsule and thereby be exposed to the acidic joint fluid which is especially efficient a leaching out inorganic lead and releasing it into the bloodstream.

In conclusion I hold multiple opinions to the level of reasonable medical certainty. First, Morgan Harris–Workman was a neurologically intact, normal individual prior to the mass shooting in which she was wounded on November 5, 2017. Second, she sustained a gunshot wound to the left leg with multiple retained fragments of shrapnel there as well as in the regions of her right shoulder and right face. Third, she had an acute transient mechanical difficulty weight bearing and walking, related to the acute injury and explained by or resulting from the local swelling and pain associated with the acute injury. That has now resolved. Fourth, approximately two and one-half months after the gunshot wound, she had onset of a subacute progressive distal neuropathy affecting the lower extremities with motor impairment greater more than sensory. The principal functional impact of the neuropathy was a gait disturbance, primarily through the consequence of the motor

impairment. Fifth, the underlying cause of this neuropathy is lead poisoning arising from the retained shrapnel. Sixth, at this point her neuropathy is chronic or static. Her original deficits have only partly resolved to the current point of mild stocking-glove sensory disturbances of limited functional impact but ongoing motor deficits of obvious and significant functional impact. She has ongoing need of various orthopedic devices and requires adaptive devices for safe automobile operation. Seventh, the long-term prognosis of the neuropathy attributable to lead poisoning from retained shrapnel is uncertain. While her present status is improved from its nadir and functionally stable in the recent year, uncertainty exist about recrudescence which could arise in the event of additional mobilization of lead from the retained shrapnel, most of concern with respect to shrapnel fragments proximate to the left knee joint. Eighth, long-term prognosis for successful, safe childbearing is poor based on the present understanding of continuing lead exposure from retained bullet shrapnel and the known mobilization of lead with the cycle of pregnancy. Accordingly, she is advised against childbearing. Ninth, she has a mild chronic encephalopathy which is also attributable to lead poisoning from the retained shrapnel of the gunshot wound. The encephalopathy affects multiple cognitive domains such as calculation, memory, auditory and visual processing and organization and is most readily demonstrable in tasks requiring attention to detail. Tenth, prognosis for the chronic encephalopathy is unclear. That she may be subject to adverse effects of subsequent epochs of lead mobilization is understood, but recent medical literature also raises the additional concern that age-related cognitive decline is more pronounced among individuals with lead exposure unrelated to whether they are subsequent episodes of lead mobilization. The latter point of information suggests that prognosis of the encephalopathy should instead be guarded.

I would be happy to address any questions regarding the above.

Sincerely,

L. Douglas Wilkerson, M.D.

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